THE EFFECT OF COMPLEX GENETIC RESISTANCE IN WHEAT ON THE VARIABILITY OF *PUCCINIA GRAMINIS* F. SP. *TRITICI*

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Synopsis

The evolution of virulence in *Puccinia graminis tritici* in Australia and New Zealand has been traced over a period of approximately 50 years. Three eras are recognized; the first when no resistant varieties were cultivated, the second when those with a single gene for resistance were common and the third in which wheats with resistance controlled by 3 genes or more have been available. Four regions of the Australia-New Zealand area are recognized and although some gene flow in the organism occurs between them, two of the four show partial isolation. Asexual recombinants and mutants of the fungus provide the variability which has enabled the rust to attack resistant varieties. As more genes are accumulated into the latter the rust adapts by gaining corresponding genes for virulence. There is strong evidence that a negative relationship exists between the numbers of genes for virulence and aggressiveness but unnecessary genes for virulence were not always lost from the population. A broadly based specific resistance coupled with genetic diversity in the cultivars, has protected the Australian wheat crop from stem rust for more than twenty years.

Introduction

There has been marked success in the control of *P. graminis* Pers. f. sp. *tritici* (Eriks. and E. Henn.) in the traditional stem rust liable areas of eastern Australia. Whereas prior to 1947–48 one-quarter to one-third of the wheat crop in northern N.S.W. was ruined by stem rust in some years, there have been no significant losses in these areas for more than two decades. This degree of control has been possible as a result of research into the genetic nature of the strains and of the resistances of wheats to them.

Three Periods of Host-Pathogen Studies

Active studies on the variability of the rust organisms were commenced by Waterhouse in 1919 and these have been sustained for 50 years. The whole period is roughly divisible into 3 eras of unequal duration. The first extends from 1919 to 1938 and is marked by the almost complete elimination of the wild type strains of stem rust which were present when Waterhouse made his initial studies (Watson, 1970). The origin of these strains remains obscure but even before wheat was grown they could have survived on susceptible endemic grasses such as *Agropyron scabrum* (R.Br.) Beauv.

The second era commenced in 1938 and extended to 1964. It was characterized by intense activity on the part of the organisms adjusting to the barriers placed in their path by the growing of stem rust resistant cultivars. The latter had been developed by plant breeders who, for want of better information, had equipped each of them, in the main, with single, dominant genes for resistance. Such a genetic mechanism in the host proved insufficient to cope with the extreme variability unexpectedly found in an organism having, in this area only sporadic access to sexual reproduction. The changes in the frequency of genes for virulence in the fungus so that they parallelled similar frequency changes in the corresponding genes for resistance in the host, have already been reported in a number of studies

(Watson, 1958; Watson and Luig, 1963, 1968). Eureka (*Sr6*), Gabo (*Sr11*), Festival and Gamenya (*Sr9b*), Mengavi (*SrTt*) and Spica (*sr17*) have all been used to demonstrate the ecological relationships between host and pathogen.

The final period which extends to the present time began in 1964, and is notable for the release of a different type of cultivar. Whereas the early rust resistant wheats had mainly single genes for resistance, farmers are now growing a number of types in which the genetic base on which resistance depends has been considerably broadened. Mendos, Gamut and Timgalen, for example, all popular in the rust liable areas, are each equipped with at least three genes for resistance to stem rust. Cultivation of these wheats alongside those with either single genes or no genes for resistanc has allowed the development of a great diversity of strains.

Geographical Areas under Study

The purpose of this paper is to report on some of the characters of the rust strains found during this half-century of study. We will attempt to trace their evolution over the last 15 years and relate the accumulation of genes for virulence in the various strains to their fitness. In doing this, reference will be made to different regions of the Australia-New Zealand area where selection pressures on the fungus are markedly dissimilar. These areas are shown in Figure 1 and are as follows:

Region 1

This region includes the summer rainfall areas of eastern Australia and is made up of the northern portion of N.S.W., the Darling Downs, the Dawson-Callide Valley and the central highlands of Queensland. The traditional rust liable parts of the Australian wheat belt are encompassed within this region. Rains following harvest are common so that over-summering of rust occurs on self-sown plants and on susceptible native grasses. The most popular varieties all have more than one gene for resistance to stem rust.

Region 2

The southern fringes of region 1 merge imperceptibly into the northern limits of region 2. Included in this large area are the wheat zones of central and southern N.S.W., Victoria, South Australia and Tasmania. The wheats grown here are susceptible to the common strains of stem rust although some have a single gene for resistance. The widespread cultivation of Heron (*Sr11*) on more than 20 per cent of the northern areas of the region has favoured strains having the corresponding gene for virulence, and the presence of Gamenya (*Sr9b*) has influenced the frequency of strain 21–2,3,7. Large areas in the southern part of this region, however, are sown to Insignia, Olympic and Pinnacle — cultivars having no genes for resistance.

Region 3

The areas of Western Australia where wheat is grown are included in region 3. They are partially isolated from 1 and 2 by 700-1,000 miles of desert. In the last 10 years the total area has been divided between wheats having single genes for resistance, e.g., Gabo, and those fully susceptible. More recently Gamenya has largely replaced Gabo but the susceptible types Insignia and Falcon are widely grown. The rust population is quite different from that of the other 3 regions and inoculum passes through the dry summer mainly in the northern coastal area of Geraldton and in the southern coastal area of Esperance growing mainly on self-sown wheat plants.

Region 4

Areas of cultivation of wheat in both the north and the south island of New Zealand are included in region 4. Some isolation from Australia is apparent but there is abundant evidence that spores are transported aerially

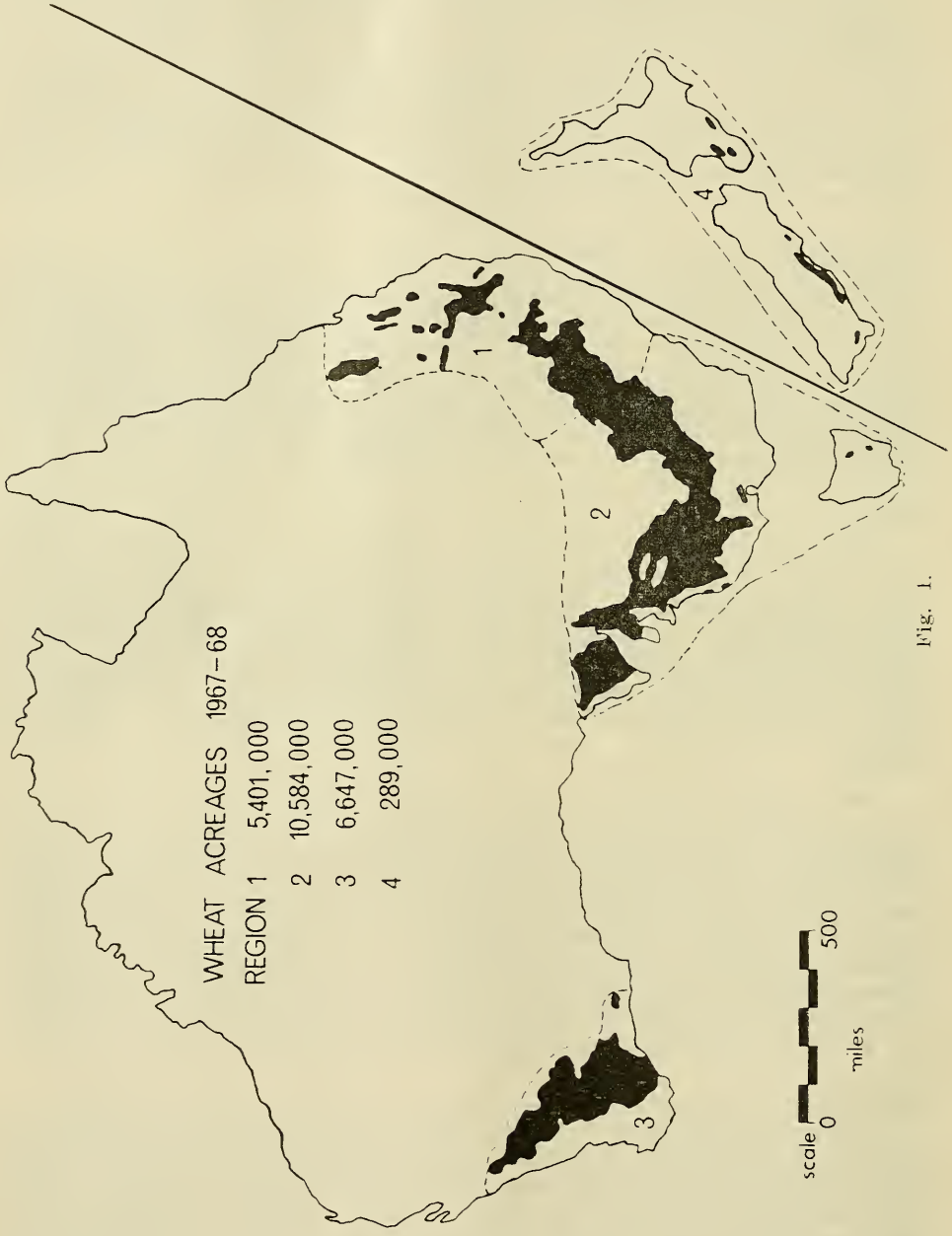

Fig. 1.

across at least 1,300 miles of water from Australia to New Zealand wheat fields (McEwan, 1966). There is, however, also clear evidence that some strains persist in New Zealand from year to year, and certain of these are very uncommon in Australia.

Designation of Stem Rust Strains

In order to clarify the description of the particular strains mentioned in this work, reference will be made to our system of classification of *P. graminis tritici* (Watson and Luig, 1963). Continuity with past records is maintained by preserving the standard race number as determined by reactions on seedlings of the 12 wheats selected by Stakman and Levine (1922). However, a more detailed description of each strain can be made when its pathogenicity on a group of supplemental differentials is known. The genes present in these latter are in sequence, 1. *Sr6*, 2. *Sr11*, 3. *Sr9b*, 4. *SrTt*, 5. *sr17*, 6. *Sr8*, 7. *Sr15*, 8. *SrW* (Webster). The procedure so successful in describing strains of *Phytophthora infestans* is incorporated so that a component strain of standard race 34 virulent on genotypes with *Sr6*, *Sr11*, *Sr9b*, and *sr17* would be 34–1,2,3,5. Festiguay, which has the Webster gene, is now included in this group of supplementals since it is susceptible to strains such as 21–2,8, 21–2,3,7,8 and 194–2,3,7,8.

Material from the field collected for identification of strains is first multiplied on Sonora W195. The resulting spores are placed onto seedlings of 4 groups of genotypes, *viz.*,

1. Useful representatives of the standard differential series: Reliance, Mindum, Acme, Einkorn and Vernal Emmer.
2. Eight stocks carrying the genes given above.
3. Genotypes which have in combination certain known resistances: Mendos (*Sr11*, *sr17*, *SrTt*), Khapstein (*Sr13*, *Sr14*), Selection 1131 (*Sr6*, *Sr9b*), Celebration (Marquillo type resistances).
4. Eagle (Falcon × *Agropyron elongatum* derivative) a genotype with resistance to all strains known in the area.

Most of the isolations are readily classifiable after one inoculation. However, separations may have to be made where two or more components together confuse the true pattern of infection. Should such separation reveal an unusual component in the mixture, it is increased and used to inoculate appropriate wheats to give the standard race number. This latter is then included in the full strain designation.

Population Changes throughout the Australia-New Zealand Area 1919–1969

The structure of the population of rust strains may be influenced by the character of the strains themselves, e.g., aggressiveness, by the physical environment and by the composition of the media (the cultivars) on which the organisms must grow. At various times at least some of these factors have influenced the prevalence of strains in Australia. The progressive elimination of the original wild type stem rusts was, as far as we know, not related to either the physical environment or to the growing of resistant cultivars although there was resistance to some of them in the popular wheats of the mid-nineteen twenties (Waterhouse, 1938). Results obtained by Waterhouse (1952) suggest that their fitness was inadequate to meet the competition from strain 126–6,7. These wild types had recessive genes for virulence on Vernal Emmer and on Einkorn which were not essential for their survival. They also had two dominant genes, one for virulence on Mindum, the other for avirulence on genotypes with *Sr5*. From our observations we suggest that both these latter fungal genes are neutral in their effect on fitness and by van der Plank's (1968) definition would be regarded as weak genes.

The competition for survival became evident in 1926 following the isolation of strain 126–6,7 (then designated form 34) late in 1925. We suggest that this strain was introduced to Australia since the only wild types from

which it could have been derived all had recessive genes for virulence on Emmer and Einkorn, and it has the dominant genes for avirulence. The studies by Waterhouse over many years show clearly how this new aggressive strain dominated the Australia-New Zealand rust flora for the succeeding 15 years. It was the strain against which Eureka and Gabo were so successful when first released in 1938 and 1943 respectively (Waterhouse, 1952).

With the passage of time and with the release of other stem rust resistant varieties, the fungi have undergone specific changes in virulence so that now, genes essential for survival and aggressiveness have become associated with genes controlling virulence. We have already reported on some aspects of this (Watson and Luig, 1966), but we now propose to give details of events in the 4 regions described above as there is no uniformity from one region to another. The changes that have occurred in these regions particularly since 1954 are as follows:

Region 1. Northern New South Wales and Queensland

Strains 126–1,6,7, 126–2,6,7, 222–2,6,7 and 222–1,2,6,7 are all closely related and have the basic phenotype of 126–6,7. We presume they have acquired by mutation, virulence on plants with either *Sr6*, *Sr11* or both. Those attacking plants with *Sr6* have the ability to do so at low (15° C.) as well as at high temperatures. As has been shown (Watson, 1958) these strains predominated until 1955 and Glenwari, Spica and Warigo, all resistant cultivars with *sr17*, were released. Celebration with Marquillo type resistance became available about the same time.

In 1954 a strain vastly different from any previously recorded from the wheat areas was isolated. This was designated 21–0. In 1948 Waterhouse had on one occasion found at high altitudes on Mount Kosciusko a rust on grass (*Agropyron velutinum* Nees.). He called this race 21 but as the culture has been lost we were unable to compare it with the material with which we have worked. The characters of 21–0 which were so different from those of the strains prevalent between 1930 and 1950 have been outlined by Watson and Luig (1966). Some of these characters, for example, avirulence on plants with *Sr5* and virulence on Mindum at low temperatures, were also present in the wild type strains and may have been inherited from them. Because these latter are not present in the wheat fields now, we have been unable to make detailed comparisons but we believe that strain 126–6,7 was aerially transported from abroad to region 3 in 1925 and strain 21–0 arrived in the same manner from abroad about 1954 and was first isolated from region 2.

Standard races 126 and 222 are very similar except for their reactions on Kubanka and Acme and for practical considerations they can be grouped together. The origin of 222 in relation to 126 is unknown but possibly they had a common ancestor. Once in Australia, however, they adjusted themselves to the resistant varieties by producing the appropriate mutants. In the absence of more aggressive rusts this 126–222 complex would doubtless have continued to vary around the genes for virulence corresponding with the resistant genes in the cultivars. However, 21–0 proved more aggressive and it overran the other components of the population, especially on hosts having no genes for resistance. We have already shown that during the course of associated growth of 21–0 and other strains, somatic recombinants were formed (Watson and Luig, 1958).

Strain 21–0, following its meteoric rise in frequency in 1955, was impeded in its progress in region 1 by the cultivation of wheats with genes *Sr11* and *sr17* and at this stage somatic hybrids could have become important since previously unrecorded types 21–2 and 34–2 were isolated. The isolation in 1957 of standard race 34 was significant. We believe it originated from a

somatic cross between a member of the 126–222 complex and race 21 since it combines characters of each. From the race 21 parent could have come virulence on Mindum, Kubanka and Acme at low temperatures, avirulence on plants with *Sr8*, *Sr15* and *sr17* and a higher infection type on plants with *Sr6* at 15° C. From the 126–222 parent, race 34 probably inherited virulence on plants with *Sr5* and a high infection type on Titan, an entry in the international wheat stem rust nursery. On Celebration race 34 gives a higher infection type than the 126–222 complex but it is lower than that given by race 21 under all but very high temperatures.

In diagram 1, beginning with 2 strains 21–0 and 34–0, we suggest that the rusts adjusted to both the physical and biological environment by producing new strains. Under the selection pressure imposed by the widespread cultivation of wheats with either *Sr11* or *sr17*, the strains of diagram 1 were among the main ones isolated. Although 34–2 was the first representative of standard race 34 to be found, presumably due to its virulence on plants with *Sr11*, we consider that 34–0 was one of the early derivatives of the somatic hybridization. Unless we assume the early existence of this latter strain it is difficult to account for the evolution of types such as 34–5, 34–6 and 34–7. The genes *Sr8* and *Sr15* were not present in commercial wheats so that virulence or avirulence on them is not related to selection.

The successful strains of region 1 required the gene for virulence on plants with *Sr11* because Gabo and derivatives also with this gene were widely grown. Once a strain had this basic ability it was free to multiply. Further variation resulting in virulence on plants with *sr17* occurred to give 21–2,5, 21–2,5,7 and 34–2,5. Strains attacking Eureka were not widespread because at no time since 1945–6 was the acreage sown to this variety extensive, nevertheless strain 21–1,2 was isolated from it in 1961–62.

Events shown in diagrams 1 and 2 were completed mainly in the second era when cultivars with single genes for rust resistance were widely grown.

Diagram 1

Variations in standard races 21 and 34 for virulence on widely cultivated varieties during 1954–1958

Previous strains	New strains virulent on varieties with	
	Sr 11	*sr 17*
	Gabo, Charter, Koda Winglen, Kendee	Glenwari, Spica, Lawrence
21–0	21–2, 21–2, 7, 21–2, 6	21–5, 21–5, 7
34–0	34–2	34–5

The third phase was initiated with the growing in 1964 of Mendos, a cultivar with three recognized genes for resistance, *SrTt*, *Sr11* and *sr17*. Prior to 1964 several strains were present and these with the acquisition of one additional ability, could have attacked Mendos. For example, strains 34–2,4 and 21–4,5 had each been found on Mengavi (*SrTt*). Although variants such as 34–2,4,5 and 21–2,4,5, virulent on Mendos, probably arose prior to 1964 they had no selective advantage and were unable to be recognized easily or to persist from year to year. They would have faced heavy competition from strains such as 21–5 and 21–4,5 on cultivars with *sr17*. Selection of strain 34–2,4,5 in a plot of Mendos close to a variety heavily rusted by strain 34–2,4 in 1964 is again strong evidence for the importance of asexual variation in these rust fungi.

Diagram 2

Variations in standard races 21 and 34 for virulence on plants with genes Sr Tt, Sr 9b, Sr 6 *and* sr 17

Prevalent strains present before 1958	New strains virulent on varieties with			
	Sr Tt	*Sr 9b*	*sr 17*	*Sr 6*
	Mengavi	Gamenya Festival Kenora	Glenwari Spica Lawrence	Eureka
21–2			21–2, 5	21–1, 2
21–2, 7		21–2, 3, 7	21–2, 5, 7	21–1, 2, 7
21–5	21–4, 5			
34–2	34–2, 4			34–1, 2

Diagram 3 summarizes stage 3 of the evolution of virulence. The variation is around 4 genes *Sr11* (2), *Sr9b* (3), *SrTt* (4) and *sr17* (5). Strain 21–2,3,7, a very common type attacking wheats with either *Sr11*, *Sr9b*, or both, gained a gene for virulence on plants with *SrTt* and was designated 21–2,3,4,7. This combined genes for virulence on the widely-grown wheats Gamenya and Mengavi but it did not become prevalent. Its derivative 21–2,3,4,5,7, on account of an additional gene for virulence on Spica (*sr17*) was able to attack Mendos, so that during the 1968–69 survey the three new strains listed in

Diagram 3

Variations in standard races 21 and 34 for virulence on Mendos

Previous strains	New strains
21–4, 5* } 21–2, 5	21–2, 4, 5
21–2, 3, 4, 7	21–2, 3, 4, 5, 7
34–2, 4	34–2, 4, 5

* It is assumed that 21–2, 4, 5 arose from 21–4, 5 and not from 21–2, 5 as the former was far more widespread.

diagram 3 comprised approximately 75 per cent. of the total inoculum present in region 1 (Figure 2). Figure 2 shows that Mendos was popular in the northern parts of region 1. Collections on it yielded one or other of the three new strains. Timgalen and Gamut, two other cultivars each with more than three genes for resistance, were widely grown but as no rust was collected on them they have been omitted in the calculations of the percentage of varieties cultivated.

During the last 15 years it has become abundantly clear that varieties with single genes for resistance to stem rust are of limited value in region 1. Genes for virulence arise in different well adapted components of standard races, and these genes correspond with genes for resistance in the host, for example, 21–5 and 34–5 are virulent on wheats with *sr17*. Provided there is no major interference with fitness such strains multiply and then undergo further changes to accumulate new abilities, e.g., strain 21–5 to 21–4,5; strain 21–2,7 to 21–2,3,7.

Region 2. Southern New South Wales, Victoria, South Australia and Tasmania

A line passing through the towns Bathurst, Orange, Dubbo, Trangie and Bourke is considered to represent the southern border of region 1. To some extent this is a very arbitrary division but resistant varieties are mainly grown north of this line. Attempts to relate virulence in the organism to

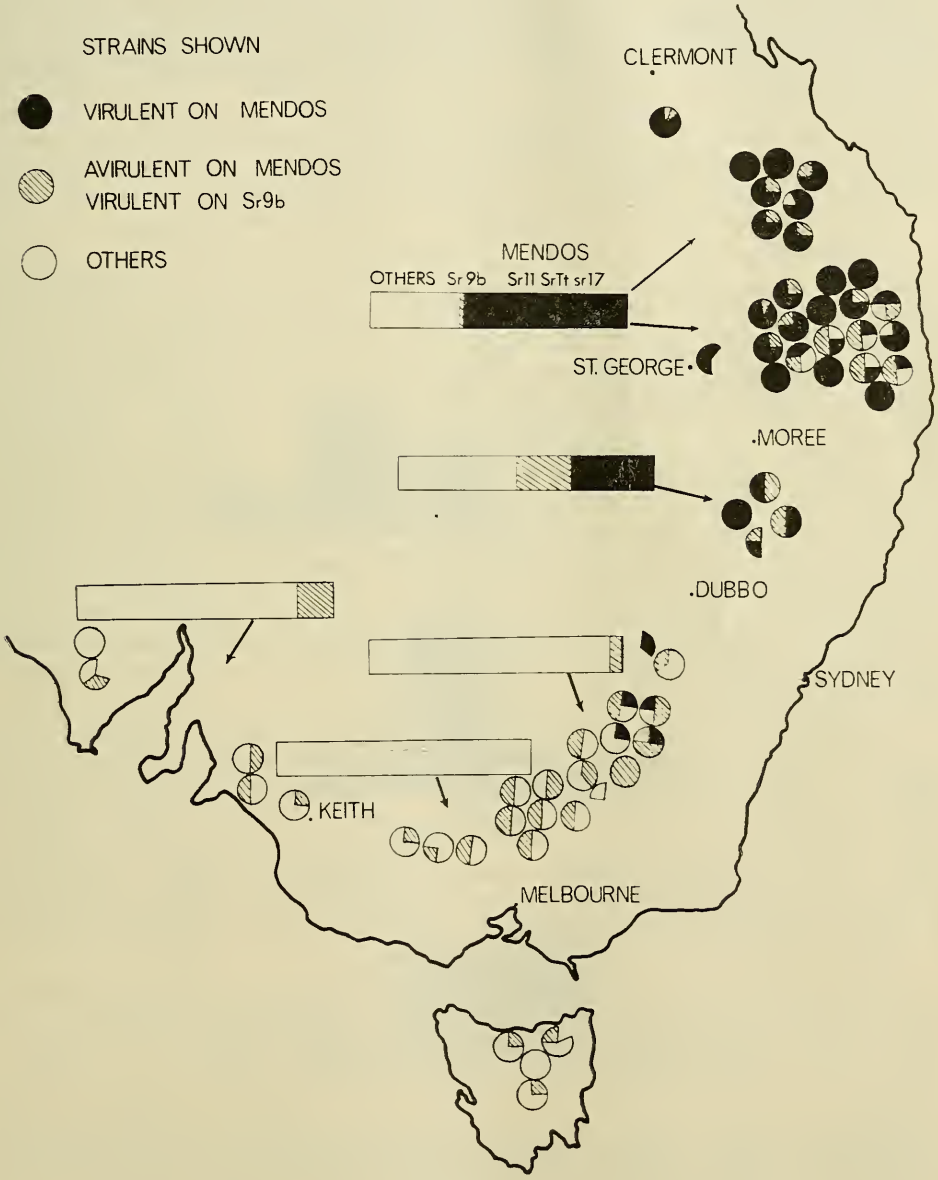

Fig. 2.

resistance in the host is complicated by the fact that there is a great diversity in the cultivars grown in region 2. Some are fully susceptible while others have a single gene for resistance. In addition, there is in certain years substantial aerial transportation of spores from region 1.

In spite of the widespread cultivation of susceptible wheats, there has been some selection pressure on the fungus and this is reflected in a slower, but nevertheless definite, increase in the factors for virulence present in the individual strains (Fig. 4). Since new strains having specific genes or combinations of genes for virulence are normally first found in region 1 it has been possible to trace their progress into other regions.

In 1954–55 strain 21–0 was first isolated in region 2 and we believe its occurrence there had no connection with events in other regions. By the end of the summer of that season it comprised 16·7 per cent of the isolates. Its aggressiveness in relation to that of other strains [126–6,7 (39·6%), 222–2,6,7 (22·9%) and 222–1,2,6,7 (12·2%)] present at the time is clearly shown in the first year. This capacity to survive and multiply was again evident in the following two seasons when 21–0 was isolated from 64·5% of the collections in 1955–6 and 78·2% in 1956–57. The increase was associated with a corresponding decline in the components of the 126–222 complex.

In 1956–57 strain 21–2, combining aggressiveness and virulence on plants with *Sr11*, arose in region 1, multiplied rapidly and became established in region 2. Both 21–2 and 34–2, however, were greatly restricted during the dry summer of 1957–58. In the following season, 1958–59, multiplication of these strains occurred in region 1 and many spores were transported to region 2. Strains 21–2 and 34–2 dominated the region and were present in 86·0% and 6·3% respectively, of the isolates. In the same year strain 21–0 was recovered only in 5·9% of the cases. Although aggressive, this strain was unable to build up when vast quantities of inoculum of other strains entered from elsewhere. In the southernmost parts of region 2 where virulence on plants with *Sr11* was unnecessary, since cultivars having this gene were grown on less than 2 per cent. of the area, 21–2 and 34–2 also predominated and constituted 90 per cent. of the isolates. Clearly the proximity of, and transportation of inoculum from one region to another, which in some years in Australia can be very important, has special significance for those experimental areas where the breeding of rust resistant wheats depends upon inoculum from elsewhere.

In region 2 the build up in frequency of strains virulent on plants with *Sr11* was largely attributable to events in region 1, but there was also some selection. This was evident on cultivars having *sr17*. In 1955–56 Glenwari (*sr17*) occupied only 6·3 per cent of the area in N.S.W., but in 1959–60 this had increased to 21·1 per cent. The increase was largely in region 2. Spica (*sr17*) was also a common cultivar. Virulence on plants with either of the genes *Sr11* or *sr17* became necessary and provided a selective advantage so that of the isolations made in 1960–61, 96·4 per cent were of strains virulent on plants with either *Sr11* or *sr17*.

The pattern of earlier years continued into the early part of the present decade. Strains originating in region 1 were transported to region 2. In 1961–62 two strains with a combination of necessary genes for virulence were isolated, *viz.*, 21–2,3,7 and 21–1,2,3,7. Both are virulent on genotypes having *Sr11* and *Sr9b* singly or in combination. Selection pressure on the organism in region 1 had been responsible for their prevalence but the cultivars concerned—Gamenya, Festival and Kenora were of little consequence in region 2. Again, proximity to inoculum was responsible for the changes in frequency of strains in region 2. Selection was not involved.

Although Gamenya was developed for region 1, it gradually moved into region 2 and with the large area sown to Heron (*Sr11*), strain 21–2,3,7 had a clear selective advantage. As shown in Fig. 2, 21–2,3,7 is a leading strain in the whole of the southern portions of region 2.

The significant differences between regions 1 and 2 are presented graphically in Fig. 2, where the distribution of selected strains in 1968–69 in the two regions is related to the area sown with susceptible cultivars or those having different types of resistance. As one moves from north to south in these two regions there is a decline in the acreage of wheats with 3 genes or more for resistance. Although regions 1 and 2 are contiguous and there is considerable gene flow in the organism from north to south, it is worthwhile to compare the changes in the levels of virulence of the strains in them since the wheats cultivated in each are basically different. In Figures 3 and 4 these changes for the 3 five-year periods in each region are illustrated graphically.

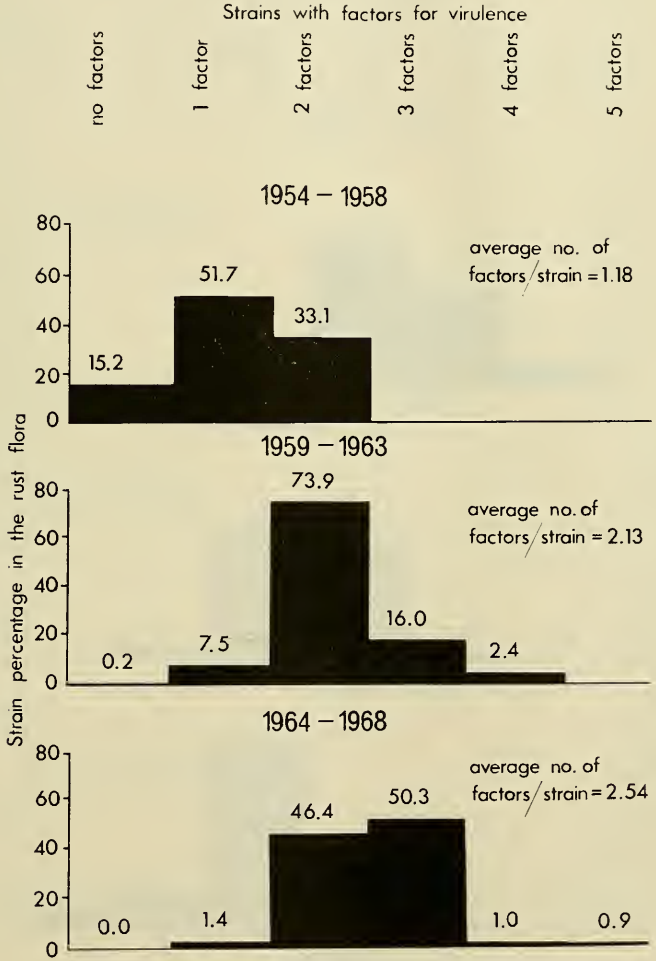

Fig. 3.

They show that there has been a progressive gain in the number of genes for virulence present in individual strains. The gain has been greater in region 1 than in 2. For region 1 the average number of virulence genes per strain over the first, second and third period was 1·46, 2·57 and 3·18 respectively. These represent gains of 76·0 and 23·7 per cent respectively. In region 2 the average number of genes for virulence per strain over the three corresponding periods were 1·18, 2·13 and 2·54, gains of 88·9 and 15·8 per cent. While for all three corresponding five-year periods the average

number of factors for virulence per strain is higher in region 1 than in region 2, the percentage increases, nevertheless, are comparable. However, if the individual categories are compared a different trend is evident (Figures 3, 4). There was no increase in the percentage of strains with four or five factors for virulence during the last five-year period in region 2 (from 2·4 to 1·9 per cent). By contrast, in region 1 the same categories increased from 4·9 to 19·2 per cent. The yearly data in Table 1 elucidates more clearly these shifts in levels of necessary factors for virulence in the pathogen.

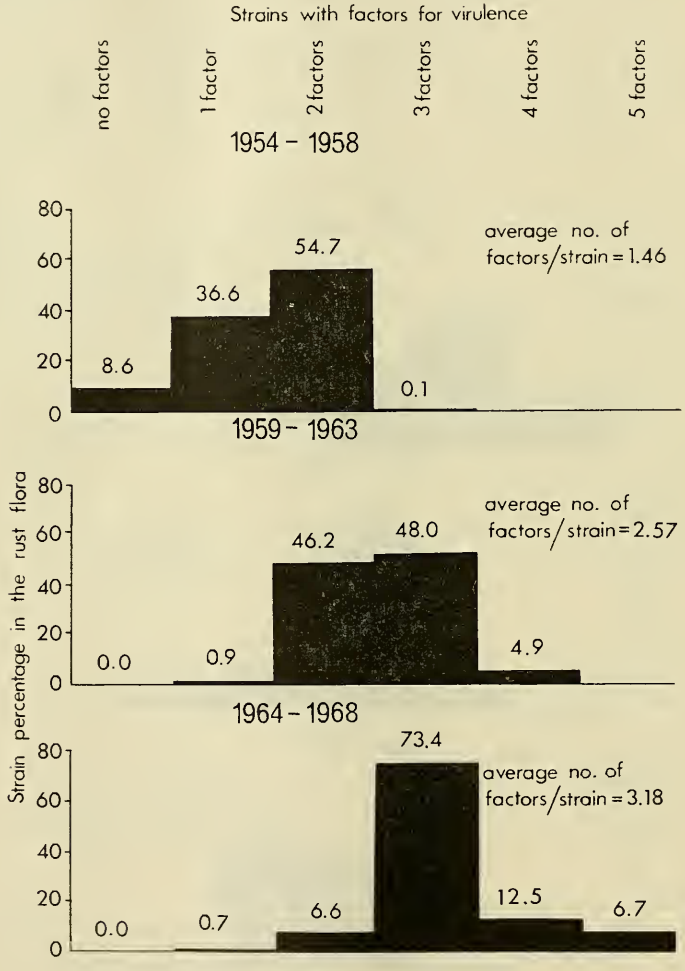

Fig. 4.

During the last season (1968–69) the percentage of strains with four and five factors for virulence in region 1 rose to 71·66 per cent while in region 2 the percentage of the same categories was 5·2. In Fig. 5 are shown the percentages of strains with three and with four or five necessary factors for virulence recovered from region 1 during the whole 15-year period. The dramatic rise in the frequency of strains with four or five genes for virulence is connected with the cultivation of Mendos (Fig. 2).

Region 3. Western Australia

Waterhouse (1952), Watson and Cass-Smith (1962) and Watson *et al.* (1966) have reported on the changes in strain prevalence in this region for the period 1939–1965. The essential point illustrated in those studies is that this region for the most part is self-contained with little opportunity for gene flow from east to west. Strain 126–6,7 was isolated first in 1925, and strain 126–1,6,7 appeared infrequently in response to the cultivation of Eureka (*Sr6*). These strains along with components of standard race 222 made up the 126–222 complex for the area.

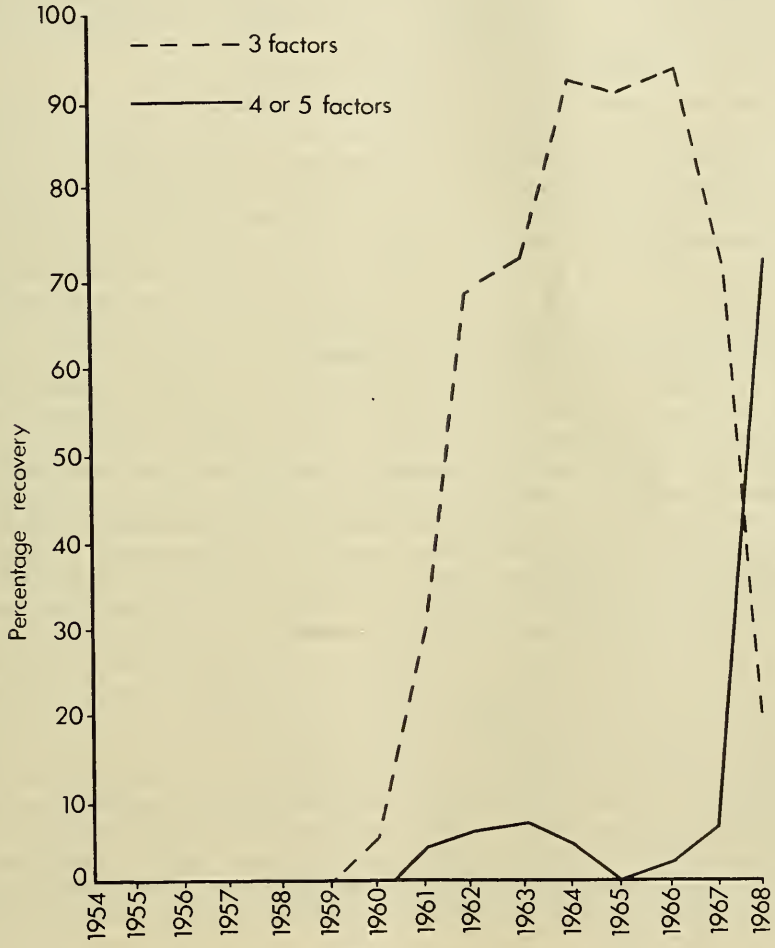

Fig. 5.

Strain 21–2, which by 1960–61 had become firmly established in regions 1 and 2, was, we presume, aerially transported from the east to region 3 and was first isolated from summer sown crops at Borden in the autumn of 1960 (Watson and Cass-Smith, 1962). The aggressive nature of this strain relative to strains of the 126–222 complex immediately showed up and within 12 months 21–2 comprised 94·1 per cent of the isolates examined from region 3. The spread was hastened by the cultivation of Gabo on 48·3 per cent of the total area sown to wheat.

The new land at Esperance on the south coast was being developed about the time of the first occurrence of strain 21–2 in region 3 and Eureka (*Sr6*) a productive and resistant wheat was the main type grown. In 1959–60 it was sown on 10·1 per cent of the total area of region 3. As indicated above, strain 21–1,2 was isolated from Eureka in the eastern states and almost simultaneously it was isolated in region 3. As gene flow into this region is rare the new strain probably occurred there as an independent mutation from 21–2. During 1962–63 and 1963–64 it increased dramatically and in the latter season was responsible for a severe epidemic. Despite the decline in the area sown to Eureka and Wongoondy, each with *Sr6,* 21–1,2, is by far the most prevalent strain in the region. From varietal statistics it would appear that this strain has not been favoured by a selective advantage due to the growing of wheats with *Sr6*. These have declined in popularity and in 1968–69 occupied less than 5 per cent of the total area.

We believe that differential fitness has developed between strains in region 3 because the pattern of survival there has been quite unlike that observed elsewhere in Australia. In regions 1 and 2, strains virulent on plants with *Sr6* have consistently declined almost to the point of disappearance since the hosts with this gene have been largely withdrawn from cultivation. By contrast in region 3, strain 21–1,2 has continued to comprise over 90 per cent of the isolates examined since 1963–64. To account for these differences we can assume that among the strains in the eastern states, genes concerned with lack of fitness are linked with the gene for virulence on plants having *Sr6* and this affects adversely their aggressiveness in the field.

The explanation for the prevalence of strain 21–1,2 in region 3 may be found by reference to the epidemic of 1963–64 when it was almost the sole contributor and overwhelmed the other strains by its specific association with Eureka, the main variety damaged. Eureka is more severely rusted than many other commercial varieties when a strain having the appropriate gene for virulence on plants with *Sr6* is involved. We suggest that variation and selection occurred among the genes controlling aggressiveness and, under the pressure of an epidemic, a type evolved in which the specific genes for virulence on genotypes with *Sr6* and *Sr11* became associated with genes for fitness. Such selection as would occur under an epidemic has not been observed elsewhere but there have been no outbreaks comparable with that in region 3 in 1963–64. If this suggestion is, in fact, the correct one, there would be little basis for assuming that an accumulation of genes for specific abilities would be parallelled by a loss of fitness.

Other standard races have been recovered from region 3 and we believe some at least have either evolved there independently by mutation or somatic recombination or have been introduced from outside Australia. Strain 34–2,4,7 is confined to region 3 and the one most closely resembling it in regions 1 and 2 is 34–2,4. The former could have arisen as a somatic hybrid but the possibility that it has been introduced from the African continent cannot be discounted.

Since wheats with broadly based resistance are not cultivated in region 3, there has been no opportunity to relate the accumulation of virulence genes with this type of resistance. Gamenya (*Sr9b*), Insignia and Falcon, each with no genes for resistance to field strains, are the leading wheats and no strain is regularly isolated virulent on plants with *Sr9b*. The data obtained over the last 15 years, however, when considered along with the genes for resistance, cannot support the proposition that genes in the fungus unnecessary for survival are lost from the population. Strains such as 21–0 could survive quite effectively in region 3 since there is a large acreage of wheat with no major gene for resistance. The fact that strain 21–1,2 has been the

leading strain for the last five years, would support the proposal that specific genes for virulence may, especially during epidemics, become associated with genes for fitness following mutation and selection and strains having such unique combinations will be aggressive and have what is seemingly more genes for virulence than necessary.

Region 4. North and South Island of New Zealand

The results obtained by Waterhouse (1952) with a number of cereal rusts would suggest that there is a unidirectional gene flow of material between Australia and New Zealand. Spore-laden winds transport the organisms across the Tasman Sea. To a large extent this results in a similarity between the rusts of regions 1, 2 and 4. When the situation from 1925 is examined it is difficult to be sure that this has always been the case, as in the early years too few collections were available from New Zealand. What little evidence there is, however, suggests that the original strains of rust present in Australia and New Zealand were much the same. Whatever the original situation, wild types in all regions had been superseded by strains such as 126–6,7 and 126–1,6,7 by 1945. Since then successive changes in regions 1 and 2 have, after a short lag period, been followed by similar changes in region 4 (McEwan, 1966). All the evidence available suggests that strains originating in region 1 may be transported to region 4 and can later be trapped there on the appropriate wheat genotype.

The two islands comprising region 4 are by no means identical in the rust flora that each maintains and it would appear that inoculum from region 1 reaches mainly the North Island. The South Island provides an environment that allows several different strains to overseason. In addition to the gene flow from regions 1 to 4, we believe that there is some evidence for independent origin of strains in region 4. The special circumstances that would facilitate such an origin are quite unknown at present as no highly rust resistant cultivars of New Zealand origin are grown to create a situation favouring the selection of specific mutants. Moreover, although members of the genus *Berberis* occur, no cases of infection by *P. graminis* have been reported (McEwan, 1966). Mutation and asexual hybridization are the most obvious explanations that can be offered at this stage to account for the variability. Once derived, the environment of New Zealand will determine the prevalence of these variants.

Stem rust has been relatively scarce in region 1 over the last three years and hence little of it has reached region 4. From the South Island of this region the identifications have been of strains with a minimum number of genes for virulence as 34–0 predominated in 1966–67 and in 1967–68, and 34–7 in 1968–69. These two strains differ in their genes for virulence on genotypes with *Sr15*, a gene which we consider to be neutral in its effects on fitness. Moreover, they differ from 21–0 and 21–7 in having the gene for virulence on genotypes with *Sr5*, a gene which also appears to be unrelated to survival.

No evidence is available from region 4 to suggest that intensive selection is operating there to favour strains having specific genes for virulence. In the North Island a small acreage of wheat is grown and Gamenya may be rusted by strains such as 21–2,3,7, and Mengavi by strains such as 21–4,5. By contrast, in the South Island susceptible wheats are grown and the prevalence of strains with genes for avirulence would favour the proposition that unnecessary genes for virulence are lost from the rust population. If this is the case, then we have in regions 3 and 4 quite distinctly different situations. Studies with *P. recondita* f. sp. *tritici* Rob. ex Desm. in region 4 somewhat parallel those reported herein since we know that the prevalent

strains are not those that are transported from regions 1 and 2 but are very similar to the original wild types reported by Waterhouse 40–50 years ago. No significant work on breeding leaf rust resistant wheats has been done in New Zealand and hence little selection has been observed in the rust population. The predominant strains continue, for the most part, to be wild types with several genes for avirulence, although we have known for a long time that prevalent strains from the Australian mainland have found their way to both islands.

Data from region 4 allows no interpretation to be made on the relation between genetically broad-based resistance and accumulation of genes for virulence. No resistant cultivars have become popular there and those that have been grown on small areas have single genes for resistance.

Evolution of Virulence

It is now well known that new strains of *P. graminis* differing in genes for virulence may arise sexually or asexually. Under Australian conditions where sexual reproduction in *P. graminis* is virtually non-existent, there has been an excellent opportunity to trace the evolution of virulence in the strains. Mention of this has already been made in the diagrams presented, but in addition there are other marker characters of various strains which are revealed only when they are inoculated on to the appropriate host genotypes.

Sexual reproduction allows the segregation and recombination of genes concerned with virulence. Genes and gene combinations which have particular selective advantage or which are combined with genes for aggressiveness, soon become frequent in the population. By contrast, asexual reproduction which only involves mutation and some type of somatic hybridization is less efficient at bringing these genetic systems together. However, when the mutant or the asexual recombinant has the appropriate genes for virulence combined with those for aggressiveness then it, like the sexual counterpart, can become a predominant strain.

Whether or not sexual reproduction is important is reflected in the diversity of the genes for virulence of strains collected in the field. Sexual reproduction is associated with large numbers of different standard races, e.g., in North America and in Europe where barberries are, or were, frequently infected by *P. graminis* there is a wide range of standard races (Stakman *et al.*, 1962); some of them, not particularly relevant for breeding work, occur with significant frequency.

Despite considerable work in Australia, South Africa, Kenya and India where the alternate host is unimportant, relatively few standard races have been recorded (Lombard, 1965; Guthrie, 1959; Pal, 1968). In such countries active breeding programmes are in progress and the organisms, lacking opportunity for sexual reproduction, show variation within standard races rather than between them. In other words, the variation is distributed around a different set of genes of the host. In Australia, for example, races 21 and 34 predominate, but over 50 different components of them have been isolated. Maximum variability will occur in areas such as North America where sexual reproduction promotes recombination for one set of genes and breeding programmes select variants having another set.

When certain standard races are prevalent in the field, asexual variants will appear from time to time in their progeny. Such variants may show virulence on wheats of the standard differential series or on those of the local supplemental series: if on the former, then the new standard races will, in the main, be those represented by single gene mutations from the parental type as shown in diagram 4 for the standard testers Einkorn and Vernal Emmer.

During the last 15 seasons strains virulent on Einkorn or Emmer have been recovered 76 times from 7,262 samples from the wheat belt of eastern Australia. Nearly all of these strains were found in region 1 (1·61 per cent of the total isolates) and with a frequency at least 10 times that for the same strains collected in region 2 (0·14 per cent of all isolates). Of the 76 isolates, 60 were virulent on Vernal Emmer, strain 116–2,3,7 being the main representative of this group.

Diagram 4

Suggested mutations in standard races 21 and 34 for virulence on Einkorn and Emmer

Prevalent field strain	New strain virulent on	
	Einkorn	Vernal Emmer
21–2	17–2	116–2
21–1, 2	17–1, 2	
21–4, 5		116–4, 5
21–2, 3, 7	17–2, 3, 7	116–2, 3, 7
21–1, 2, 3, 7	17–1, 2, 3, 7	
34–2		40–2
34–2, 4, 5		40–2, 4, 5

No resistant wheats have been developed in Australia with the resistance of either Vernal Emmer or Einkorn and at no stage has selection pressure been placed on the organism at loci concerned with virulence on them. In the terminology of Van der Plank, these host genes would be "weak", giving little protection because some strains in the rust population have the corresponding genes for virulence even before the commencement of a breeding programme. Furthermore, there would be little selection against such strains once they become more prevalent due to the presence of the specific hosts. Other "weak" genes which could be mentioned in this context are *Sr5* in Reliance, *Sr15* in Norka and *Sr8* in Mentana. The mutation rate in the rust towards virulence on plants with *Sr15* is particularly high and many strains have counterparts virulent and avirulent on seedlings with this gene. This applies to a much lesser degree in the case of *Sr5*, although a strain, 34–1,2,3,7, which proved to be almost identical with strain 21–1,2,3,7, from which it originated in the glasshouse, is evidence of a reasonably high mutation rate. Strain 34–7, which has now become the most widespread rust in New Zealand, appears to be a mutation from 21–7 and the replacement of a dominant gene for avirulence by a recessive gene corresponding to the *Sr5* locus even suggests that there may be some selective advantage.

The manner in which strains have evolved around genotypes with *Sr15* suggests that the corresponding rust gene may represent an extreme case of an association between fitness and virulence. Variation about this gene may take place in a genotype that is already aggressive, so that only in some instances does a change for virulence in either direction make the strain more aggressive. The low frequency of the gene for avirulence on genotypes with *Sr15* in the United States, as found in a limited study by one of us (I.A.W.), would also suggest that in that area virulence would have no adverse effect on fitness.

Much of the information on the evolution of virulence comes from a close study of the components of race 21 and 34 in all 4 regions. Strain 21–0 reached New Zealand in 1956–7. On the South Island where the rust may persist from season to season (McEwan, 1966), strain 21–7 is common, and has arisen presumably by mutation. Moreover, in association with it, we

have found 34–7 to be common. In contrast to the 34–7 of mainland Australia, 34–7 from New Zealand gives a high infection type on Celebration which is characteristic of its 21–7 parent.

Further evidence has recently revealed other differences between standard races 21 and 34 and their components. It had its origin in the field behaviour of Mendos in 1968 in region 1 where it was infected at several sites. Studies showed that the strains involved were mostly 21–2,4,5, 21–2,3,4,5,7 and 34–2,4,5 which, by their formulae, would be expected to be virulent on seedlings of Mendos (*Sr11*, *SrTt*, *sr17*). To the first two of these strains, however, Mendos seedlings give an "X" infection type and we believe that a gene other than the three listed provides some protection to both seedlings and adult plants. By contrast, strain 34–2,4,5 and 40–2,4,5 derived from it both give "3" type infections on Mendos seedlings. This suggests that if a fourth gene is present in this cultivar it is ineffective against strain 34–2,4,5.

Two entries from the International Rust Nursery IRN67–197 (Barleta Benvenuto) and IRN67–317 (Titan) have reacted similarly to all components of standard races 21 and exhibit "X–" infection types. To all components of standard races 34 and 126 infection type "3" is typical. It is not yet known whether they share a common gene with Mendos, but these 2 entries are very useful in tracing the origin of strains belonging to standard races 21 (or 17, 116, 194) and 34 (or 40). On the standard set these are differentiated on Reliance (*Sr5*) and, as mentioned earlier, on the Australian variety Celebration there is also a distinct difference in infection types. Thus, a mutation for pathogenicity on either of these two latter varieties would make it uncertain as to which group of standard races the original strain belonged. Since Summit (*Sr5*) has been released in region 2, mutations in standard race 21 could be expected. We have now tested many different strains on Barleta Benvenuto and Titan and in all cases infection types conformed to those expected. These infection types, together with those recorded previously on Celebration, indicate the origin of such strains. *Sr15*, on the other hand, is useless in this connection as many strains can be subdivided on it. We also assume a low mutation rate in the fungus of the genes for infection types on Barleta Benvenuto, Titan and Celebration.

The evolution of virulence around the genes *Sr5*, *Sr6*, *Sr8*, *Sr9b*, *Sr11*, *sr17* and *SrTt* have been approximately along pathways that could have been predicted. In the case of the resistance from Webster, which is available in the commercial variety Festiguay, expectation has not been realized. Shortly after the latter was released in 1963 when strain 21–2 was prevalent, we isolated strain 21–2,8 virulent on both Festiguay and Webster. This we assumed to be a simple mutation as it resembled the putative parent 21–2 in all other respects. During the last 12 months, however, other material has been studied also virulent on Festiguay but its origin cannot be clearly explained at present.

The adult plant resistance of Webster is conditioned mainly by a single factor which also operates in the seedling stage to give plants with "2+" and "3c" infection types. Under field conditions when temperatures exceed about 27° C.. Festiguay may show considerable rust development even to strains avirulent on it. The pustules, although restricted in size, would allow strong competition against any mutant fully virulent on plants having this type of resistance. This competitive effect may have helped to maintain the resistance of Festiguay over a number of years, even though only a single factor was concerned. This may even represent an advantage of the semi-resistant over the immune reaction type.

Two strains were isolated in 1968–69 to which Festiguay is fully susceptible. An examination of the infection types of the first of these shows it

to conform to those produced by either standard races 194 or 279 which are very similar. These latter are characterized by low infection types on both Kubanka and Acme, but otherwise are very close to race 21. At the present time, because Kubanka and Acme give identical infection types with this Festiguay strain we prefer to designate it 194–2,3,7,8.

When examining mutant strains in relation to their origin it has not been usual to find that the acquisition of a new gene for virulence is accompanied by a loss of other genes for this character. For example, a gain in virulence on Festiguay would not be expected to be associated with a loss of virulence on Kubanka and Acme.

This Festiguay attacking strain gives low infection types on the latter two wheats as well as on Celebration. The closest putative parent would be 21–2,3,7, but the latter shows a high infection type on Kubanka, Acme and Celebration. It has been difficult to use the former two of these three wheats to trace evolution of virulence since there are a small number of components within standard races 21 and 34 which on Kubanka and Acme show lower infection types but are not avirulent. Since the origin of the first of these Festiguay strains is not clear we cannot overlook the possibility that it has been introduced into the country.

Standard races 194 and 279 are both known to occur on the African continent but at present we have been unable to determine the infection types when these overseas isolates are inoculated onto plants with *Sr11, Sr9b* and *Sr15*. When this is done there may be some additional evidence to suggest the areas from which this Festiguay strain could have originated.

The second strain on Festiguay has been designated 21–2,3,7,8 and it might at first appear to be a mutation from the common 21–2,3,7. However, on Mentana (*Sr8*) the former produces a "2+" infection type in contrast to the "2–" or "3+" types normally observed with avirulence or virulence respectively on this differential. In both of these new strains the gain in virulence on Festiguay has been associated with a change in pathogenicity on other unrelated genotypes. No selection pressure which required the strains to gain or lose genes concerned in the infection types on Kubanka, Acme, Celebration or plants with *Sr8* has been applied but there is clear evidence that if these strains have evolved from local parental strains, changes on Festiguay have not been independent of other changes. These differences between parental types and their variant progeny could be expected following asexual hybridization rather than mutation, but so far we have made no attempt to recover these new types from mixtures of existing strains.

Discussion

The data presented indicates clearly that the use of a broadly based resistance in the wheat plant has not given permanent control of stem rust. However, the manipulation of genetic factors in various combinations has resulted in a degree of diversity in the germplasm that has greatly reduced losses in the traditional rust liable areas. Regardless of the genetic nature of the resistance, as long as susceptible hosts are grown new pathogenic strains of the rust organism will arise. Recombination of the genes for virulence under sexual reproduction will give rise to dramatic changes in the phenotype of the rusts. Less spectacular, but nevertheless well adapted and destructive variants, will arise from mutational and somatic hybridization processes. In Australia, strains arising asexually have caused the greatest concern.

For the most part the Australia-New Zealand areas are well isolated from other wheat areas of the world. The assumption that this is complete

must be questioned since evidence is accumulating that from time to time spores from wheat fields outside the south-west Pacific may reach this region. Satisfactory explanations for the unpredicted arrival of the standard race involved in the 126–222 complex in 1925–26, of strain 21–0 in 1954–55, and of 194 (or 279) in 1968–69 are not forthcoming. It is tempting to propose that each had its origin in the areas extending from South Africa to India but unfortunately there is inadequate data on the nature of the components of the standard races present in these areas. Only by global studies of the genes for virulence will this problem ultimately be satisfactorily studied.

Even without the influx of new fungal germplasm there has been extraordinary variation in *P. graminis* during the past 50 years. Variants have evolved which are adapted particularly to cultivars having single genes for resistance. As we have shown (see summary—Macindoe and Walkden-Brown, 1968) *Sr6* became ineffective in 1942, *Sr11* in 1948, Marquillo in 1954, *sr17* in 1959, *Sr9b* in 1960 and *SrTt* in 1961. We know that the single gene in Webster is now ineffective. Some single genes may nevertheless be highly effective in this area against all strains. The gene from *Agropyron elongatum* present in Eagle (AR–Falcon 28A), *Sr13* from Khapstein, a gene other than *SrTt* in *Triticum timopheevi* Zhuk., and the adult plant resistance of Hopps are all useful sources of resistance in this area. There are probably several more which have not yet been isolated.

The evidence in favour of combined resistance is encouraging but leaves no grounds for complacency. Breeding work in Australia has aimed to broaden the genetic base but this has not always been carried to completion. Tight linkage in repulsion between *Lr3* and *Sr11* has prevented the ready incorporation of *Sr11* into Gamenya and Mengavi. Again, close linkage or allelism on chromosome 2B of the genes *Sr9b* and *SrTt* has so far prevented this useful combination of genes.

While breeders are incorporating more and more genes for resistance into commercial wheats, new combinations of the corresponding genes for virulence are arising by chance and are found in comprehensive surveys. Four Australian cultivars which carry more than one gene for resistance are now susceptible, and this may suggest that combinations provide little advantage. However, Robin and Raven, which have *Sr11* and *Sr9b* combined, were released at a time when strains capable of attacking plants with this combination were already widespread. The third variety Moora was developed in Western Australia and it combines the genes *Sr6* and *Sr9b*. At the time of its release in 1958 both genes were highly effective. It has remained resistant in region 3 although *Sr6* alone is ineffective due to the prevalence of strain 21–1,2. Protection is provided by *Sr9b*. We believe that conditions in region 3 have not been appropriate to assess the particular combination *Sr6 Sr9b* under an epiphytotic caused by 21–1,2. In region 1, by contrast, Selection 1131 (*Sr6Sr9b*) under a minor outbreak of 21–2,3,7 showed pustules of a new rust 21–1,2,3,7 (Watson and Luig, 1963). We have assumed it was a mutant. The fourth cultivar with a combination of genes for resistance is Mendos. In 1964, at the time of its release (Table 1), strains which had combined genes for virulence corresponding with any two resistance genes of Mendos were in the field. Strains 21–4,5 and 34–2,4 attacked plants having respectively the combinations *SrTt*, *sr17* and *Sr11 SrTt*. It required only a single gene mutation to obtain the virulence formula 2,4,5 appropriate for Mendos and in 1964 strain 34–2,4,5 was isolated on Mendos by one of us (N.H.L.) in a plot alongside Gabo and Mengavi heavily rusted by 34–2,4. Environmental conditions prevented the multiplication of 34–2,4,5 but it was isolated again in 1967, and by this time, too, 21–4,5 and/or 21–2,5 had mutated to 21–2,4,5. The distribution of these and other strains in 1968

has been graphically presented in Figure 2 where strains 21–2,4,5, 34–2,4,5 and 21–2,3,4,5.7 (a probable mutation from 21–2,3,4,7) contributed substantially to the inoculum of region 1.

The resistant varieties cultivated widely in region 1 in addition to Mendos, are Timgalen, Gamut and Festiguay. Festiguay is now susceptible to two strains and, because it has a single major gene, need not be discussed further. Timgalen and Gamut, each with at least four resistance genes, have so far withstood all atacks from rust in the field, although laboratory cultures have been derived to which Timgalen is susceptible. The presence of the gene, tentatively called *SrGt,* protects Gamut in the field against rare strains such as 21–1,2,3,7. In order for these varieties to become susceptible, changes at more than one locus in the fungus will be necessary and to this extent the value of the broad base has been established.

The maintenance of the resistance of Gamut and Timgalen may also have been helped if, as suggested by Van der Plank (1968), there is a negative association between fitness and a broad spectrum of genes for virulence. Gamut has the genotype *Sr6 Sr9b Sr11 SrGt* and the strain 21–1,2,3,7 would require only one additional ability in order to attack Gamut. However, this strain, although recorded on several occasions, has poor survival ability in the field, a character we believe is associated with the homozygous recessive condition of the gene for virulence on plants with *Sr6.* Timgalen with the genes *Sr5 Sr6 Sr8 SrTt* and *srT* has, we believe, survived for the reason that it has a broadly based resistance and that strains which have combined several of the genes for virulence necessary to attack Timgalen persist in low frequency, if at all, from season to season.

The work presented here cannot fully support the proposals advanced by Van der Plank (1963, 1968). We believe that genes for virulence are for the most part not concerned in aggressiveness although, when first produced, being recessive, they may show differential survival and would thus tend to be associated with reduced fitness as reported by Watson (1958) and Watson and Luig (1963). Survival of strains 21–1,2 and 34–1,2 both virulent on plants with *Sr6* has been poor in eastern Australia, although if cultivars with *Sr6* alone were widely cultivated both strains would be important. In regions 1 and 2 neither strain has been concerned in a major epidemic and it is difficult to isolate them on a host other than that for which they are specific. Results in region 3 on the other hand suggest that 21–1,2 is particularly well adapted on cultivars not specific for it as well as on those with *Sr6.* It is significant, we believe, that, since its original isolation in 1961, it has been involved in a major epidemic in which it was essentially the only competitor. On this evidence we predict that if strains such as 34–1,2,3,4,5,6,7 (a laboratory strain and a poor competitor) could be built up in the field and could be encouraged as the sole contributor to cause an epiphytotic, variants with enhanced fitness would arise without loss of genes for virulence. Already we have shown that in mutants for virulence, produced by E.M.S. treatment (Luig, 1967), there is a wide range of types for sporulation times, ability to break the epidermis and for other characters important in survival. From such populations it has been possible to select cultures having good survival ability in the glasshouse combined with the necessary genes for virulence. Under field conditions, natural selection will accomplish the same result.

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TABLE 1

Summary of the number of isolations of different strains of Puccinia graminis tritici *grouped according to areas and five-year periods from 1954–55 to 1968–69*

	21–0	21–2	21–2, 6	34–2	34–4	126–6, 7	126–1, 6, 7	126–2, 6, 7	222–6, 7	222–2, 6, 7	222–1, 2, 6, 7	222–1, 2, 4, 6, 7	Total
1954–55													
Q.	3	—	—	—	—	6	—	1	—	14	3	—	27
N. N.S.W. ..	9	—	—	—	—	34	6	3	3	28	14	1	98
S. N.S.W. ..	22	—	—	—	—	58	2	1	6	35	18	—	142
Vic.	8	—	—	—	—	12	6	—	1	9	5	—	41
S.A.	1	—	—	—	—	1	—	—	1	—	—	—	3
Tas.	1	—	—	—	—	1	—	—	—	—	—	—	2
W.A.	—	—	—	—	—	4	—	—	—	—	—	—	4
N.Z.	—	—	—	—	—	—	—	—	—	—	—	—	—
Total ..	44	—	—	—	—	116	14	5	11	86	40	1	317
1955–56													
Q.	19	—	—	—	—	5	—	2	—	2	—	—	28
N. N.S.W. ..	62	—	—	—	—	18	2	16	—	26	12	—	136
S. N.S.W. ..	121	—	—	—	—	27	3	17	—	7	—	—	175
Vic.	13	—	—	—	—	5	—	1	—	2	—	—	21
S.A.	11	—	—	—	—	11	—	3	—	2	—	—	27
Tas.	8	—	—	—	—	6	3	—	—	—	1	—	18
W.A.	—	—	—	—	—	12	—	—	—	—	—	—	12
N.Z.	—	—	—	—	—	—	—	—	—	—	—	—	—
Total ..	234	—	—	—	—	84	8	39	—	39	13	—	417
1956–57													
Q.	11	18	—	—	—	—	—	5	—	1	—	—	35
N. N.S.W. ..	45	34	—	—	—	—	—	15	—	4	—	—	98
S. N.S.W. ..	86	8	—	—	1	—	—	8	—	2	—	—	105
Vic.	24	4	—	—	—	3	—	1	—	—	—	—	32
S.A.	11	—	—	—	—	—	—	4	—	2	—	—	17
Tas.	8	—	—	—	1	—	—	2	—	—	—	—	11
W.A.	—	—	—	—	—	8	—	—	—	—	—	—	8
N.Z.	7	—	—	—	—	4	—	—	—	—	—	—	11
Total ..	192	64	—	—	2	15	—	35	—	9	—	—	317
1957–58													
Q.	1	26	—	—	—	—	—	—	—	—	—	—	27
N. N.S.W. ..	—	54	—	2	—	1	—	3	—	1	—	—	61
S. N.S.W. ..	7	1	—	—	—	—	—	—	—	—	—	—	8
Vic.	11	2	—	—	—	—	—	—	—	—	—	—	13
S.A.	—	—	—	—	—	—	—	—	—	—	—	—	—
Tas.	—	2	—	—	—	—	—	—	—	—	—	—	2
W.A.	—	—	—	—	—	3	—	—	—	—	—	—	3
N.Z.	—	—	—	—	—	—	—	—	—	—	—	—	—
Total ..	19	85	—	2	—	4	—	3	—	1	—	—	114
1958–59													
Q.	4	56	—	4	1	—	1	—	—	—	—	—	66
N. N.S.W. ..	2	192	1	22	—	2	1	5	—	—	—	—	225
S. N.S.W. ..	10	180	—	12	—	3	2	—	—	—	—	—	207
Vic.	3	26	—	1	—	—	—	—	—	—	—	—	30
S.A.	—	15	—	2	—	—	—	—	—	—	—	—	17
Tas.	3	13	—	2	—	—	—	—	—	—	—	—	18
W.A.	—	—	—	—	—	6	—	—	11	—	—	—	17
N.Z.	—	2	—	—	—	1	1	—	—	—	—	—	4
Total ..	22	484	1	43	1	12	5	5	11	—	—	—	584
Grand total (five years)	511	633	1	45	3	231	27	87	22	135	53	1	1,749

TABLE 1 (continued)

	17–2	17–1, 2	17–2, 3, 7	21–0	21–2	21–5	21–6	21–1, 2	21–2, 5	21–2, 6	21–4, 5	21–5, 7	21–1, 2, 6	21–1, 2, 7	21–2, 3, 6	21–2, 3, 7	21–1, 2, 3, 7	21–2, 3, 4, 7	34–0	34–2	34–4	34–5	34–6	34–1, 2	34–2, 4	34–2, 5	34–2, 7	34–2, 4, 7	40–2	116–2	116–2, 3, 7	126–6, 7	126–1, 6, 7	126–2, 6, 7	222–6, 7	Total
1959–60					35	10														5					1										51
Q.					146	8														67															230
N. N.S.W.				9	184	22														16											2				262
S. N.S.W.				36	25	1														5															34
Vic.				3	37															8															51
S.A.				6	9															2															14
Tas.				3	1																												36	37
W.A.					27															3											54				95
N.Z.				11																														
Total				68	464	41														106					1							56		2	36	774
1960–61					16	11										4				1															32
Q.					45	5														21					1										73
N. N.S.W.				1	57	16				1										9															83
S. N.S.W.					14	1														2															19
Vic.				2	19															4															23
S.A.				3	9	1																													13
Tas.					188																										12			3	203
W.A.				1	25																														26
N.Z.																																			
Total				7	373	34				1						4				37					1							12			3	472
1961–62					55	40		1								52	11			8					5						2		1		175
Q.					129	44		5	2							86	16			124					26				1		8		1		444
N. N.S.W.	2				59	26		1								16	6			20															130
S. N.S.W.				2	13															1															16
Vic.				2	5	1														2															9
S.A.				1	6	2				1						1																			13
Tas.				3	106			8																							2				116
W.A.					17	2										1			7	2															31
N.Z.				2																															
Total	2			10	390	115		15	2	1						156	33		7	157					31				1		10	2	2		934
1962–63					6	22		5								62	8			6				109					2	7					227
Q.					63	34		60	3	1						147	50			85			10	171	5			1		12		2			651
N. N.S.W.	1	3	2	1	38	33		7	2							24	14			18			1	24											161
S. N.S.W.					14			1	1							1																			17
Vic.					4			2												4															10
S.A.					15	5																													20
Tas.					10			54																		3									69
W.A.		2			20	25										5	1			1					1										60
N.Z.				7																															
Total	1	5	2	8	170	119		129	6	1						239	73			114			11	304	6	3		1	2	19		2			1,215
1963–64				3	2	6			1		13			3		37	5			2			2	49				2		4					129
Q.				2	31	23			3		13			45		57	23	1	2	16	1	2	12	89				2		6					328
N. N.S.W.				6	38	30		31	2						1	23	6		1	26			2	15											182
S. N.S.W.				6	4	5		3								3	1			11				1											34
Vic.		1		3	3			10											1	9			2	1											30
S.A.				4	7		1	3								2								1				1							19
Tas.		2			22			273				1	1	1										1		1	3			1					305
W.A.				22	22	23													3	1		1		4				1							77
N.Z.																																			
Total		3		46	129	87	1	320	6		26	1	1	49	1	122	35	1	7	65	1	3	1	18	160		1	4	5		11					1,104
Grand total (five years)	3	8	2	139	1,526	396	1	464	14	3	26	1	1	49	1	521	141	1	14	479	1	3	1	29	497	6	4	4	7	2	40	70	4	2	39	4,499

TABLE 1 (continued)

	17–1, 2	17–2, 3, 7	17–1, 2, 3, 7	21–0	21–2	21–5	21–7	21–1, 2	21–2, 5	21–2, 6	21–2, 7	21–2, 8	21–4, 5	21–5, 7	21–1, 2, 7	21–2, 3, 4	21–2, 3, 7	21–2, 4, 5	21–2, 5, 7	21–5, 6, 7	21–1, 2, 3, 7	21–1, 2, 5, 7	21–2, 3, 4, 7	21–2, 3, 5, 7	21–2, 3, 7, 8	21–2, 3, 4, 5, 7	21–2, 3, 4, 5, 6, 7	34–0	34–2	34–5	34–7	34–1, 2	34–2, 4	34–2, 5	34–2, 7	34–(4), 5	34–5, 7	34–1, 2, 7	34–2, 3, 7	34–2, 4, 5	34–2, 4, 7	34–2, 5, 7	34–1, 2, 3, 7	40–2, 4, 5	116–4, 5	116–2, 3, 7	194–2, 3, 7, 8	Total
1964–65																																															
Q.						4							72				54				3								1	2			29												2		167
N. N.S.W.		2		1	2	3		20	2		1	1	63	2		1	88				4								5			9	82	1									1	1		298
S. N.S.W.				6	11	2			2			1	8	1			24												5				5	2	1											68
Vic.				1	3	1								1															4						1											11
S.A.					1			1																					17									2							21
Tas.					2	2							1	1															3																9
W.A.			1					66																														6							73
N.Z.				9	4	9					2		21	14			11		1	1	1	1							1			6				1										83
Total		2	1	17	23	21		87	4		3	2	165	19		1	177		1	1	8	1						1	35	2	1	9	122	3	2		1		9	8				1	3		730
1965–66																																														
Q.								1					49				35			5								1		2		3													96
N. N.S.W.				1									3				8												1			2				1									16
S. N.S.W.				1	19	1							11				6												8			2											1		50
Vic.																	1																												1
S.A.																																														—
Tas.																																														—
W.A.								91						1																									1							93
N.Z.					4	3	14				1		1	24			1												11																60
Total				2	23	4	14	92			1		64	25			51			5								12	9	2		7				1	1			1	1			1		316
1966–67																																														
Q.				3	1	1			2		1		44				52		3	3										3		1													114
N. N.S.W.		1	1		3		1		3				45		1		113		1				1						2			1									1				174
S. N.S.W.		1		1	24			1	4				50	15			115		2	1			1						46	1		2													264
Vic.					6				1	1			5				6												5																24
S.A.																																														—
Tas.					1																								1																	2
W.A.	1				1			73																														1				1			78
N.Z.				3	1								3	8			5	1										12	1																34
Total	1	2	1	7	37	1	1	74	10	1	1		147	23	1		291	1	6	4			2					12	55	5		4				1	1			1						690
1967–68																																														
Q.				2	5	7			4				37				35	1											1	6		2						4							104
N. N.S.W.					3		1						5				21	1					2	1					1									2							37
S. N.S.W.		1			3	2			4				7				21												17	1															56
Vic.					1																								1																2
S.A.																	1												7																8
Tas.																																														—
W.A.							8																																							8
N.Z.				2										3			3						1		1		14	1											1						26
Total		1		4	12	9	1	8	8				49	3			81	2					3	1		1		14	28	7		2						6			1				241
1968–69																																														
Q.		1			1	17			3		1		25	1			17	62								85	1			2								62				2	6		1	287
N. N.S.W.													1				14	3								3												6							27
S. N.S.W.					39				15		1		6				35	3							1	7			13		2		2				4	3							131
Vic.					19				3		1		1				15												10				1												50
S.A.					20												17												14																51
Tas.					26	2			3		1		2				3												1							1								39
W.A.					1		6																																							7
N.Z.					3		2		1				1	1			3		1											4	18															34
Total		1			109	19	2	6	25		4		36	2			104	68	1						1	95	1		38	6	20			3		1			4	71				2	6		1	626
Grand total (five years)	1	6	2	30	204	54	18	267	47	1	9	2	461	72	1	1	704	71	8	10	8	1	5	1	1	96	1	39	165	22	21	9	135	6	2	1	2	1	4	86	10	2	1	2	7	5	1	2,603